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Editorial

In this issue of The Veterinary Journal we conclude the publication of an article on Coccidiosis. Although the work has been carried out in the guinea pig, many of the findings can be applied equally to other animals in which coccidiosis is an important disease. This work is an example of the detailed, painstaking study which must be made in order that the fundamental knowledge of the behaviour of a parasite may be ascertained, for without such information it cannot be hoped adequately to control the parasite and the diseases arising from its action.

These remarks apply to all research work. It sometimes transpires that "short cuts" are taken and in some instances little harm results from such an action: once the general relationship of a micro-organism or a parasite to a disease has been established methods of control can sometimes be evolved. On the other hand, there are examples of errors arising from the application

of methods of control which are based on too little fundamental research into the problem. It is necessary therefore to link up all the findings of the research worker with the observations of those acquainted with the naturally occurring disease and to apply the results of the detailed research work in the evolving of a satisfactory method of dealing with the control of the condition.

We also publish in this issue an article on fowl cholera. Fowl cholera presents many problems both to the research worker and to the poultry-keeper, among which one of the most important is its occurrence in individual fowls or in a limited number of them. We may be apt to consider fowl cholera as a disease which occurs as an epi- or en-zootic, but there also exists the chronic form of the disease in which its presence is very definitely limited in extent. The chronically affected fowls or the "carrier" fowl must, however, always be considered important, for doubtless its presence in a flock keeps alive the infection which, given suitable conditions, can and does spread, sometimes to an alarming extent.

In this issue appears also the final instalment of the article on A.R.P. for animals. We referred to this subject last month. We would again point out the highly important nature of this article and we recommend its perusal by all veterinary surgeons who may be called upon to deal with casualties in animals due to enemy action. It cannot be emphasised too strongly that we, as members of the veterinary profession, must be prepared to play an important part in dealing with such casualties when they occur. The animal owners throughout the country are relying on the profession, not only for assistance with their injured animals, but also for guidance in minimising the risk of injury. It is possible, as shown in the published article, to adopt certain methods of protection for animals, and it is the veterinary profession who must be in the position to give the necessary advice.

General Articles

AIR RAID PRECAUTIONS FOR ANIMALS
(Continued)

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IT is the purpose of this article to deal with A.R.P. as primarily affecting animals. I feel, however, that it would be well to touch upon first-aid measures as applied to man. Human casualties are bound to occur and a veterinary surgeon should be able to give skilled assistance in an emergency.

FIRST-AID TREATMENT FOR HUMAN CASUALTIES.—Although the action of blister gases is delayed, first-aid treatment must be applied quickly. When a small area of the skin is splashed with liquid mustard gas first-aid treatment must be applied within five minutes if harmful effects are to be averted. If treatment is delayed beyond this time some of the harmful effects may be minimised, although irritation, redness and more or less severe inflammation of the skin will result, depending upon the period which has elapsed between contamination and the application of treatment. Mustard and lewisite are both soluble in oils and spirits. If a drop of liquid blister gas falls on the bare skin it can be removed with a swab lightly moistened with petrol, paraffin or other solvent. But this must be done immediately. In using a solvent to remove liquid contamination it is very important that not too much of the solvent should be used. A swab of cotton wool is soaked in the solvent and then well squeezed so that there is no excess of liquid. The swab is then lightly pressed over the contaminated part. The process should be repeated once or twice with fresh swabs. Used swabs should be placed in covered containers until they can be burnt or otherwise destroyed, for the solvent does not destroy the blister gas but merely acts as a dilutant. If an excessive amount of the solvent is used it may run over a large area of the skin, carrying with it the blister gas in solution and thus spread contamination to the surrounding parts. For the same reason, if a fairly large area is to be contaminated by swabbing it is best to start at the edge and swab towards the centre.

The usual method of first-aid treatment for small areas of the skin is the use of a bleach preparation which decomposes the liquid. Such treatment may be applied effectively after slightly longer delay than when a solvent is used. The bleach preparations used are bleach paste, bleach ointment and bleach cream. Bleach paste is a mixture of bleaching powder and water, and whilst its use is recommended in the case of animals, it is more irritant to the

human skin than either bleach ointment or bleach cream. Bleach ointment is a mixture of hydroscopic bleaching powder with a white mineral jelly base. Bleach cream is a mixture of bleaching powder and vanishing cream. Bleach ointment or cream should be applied not less than five minutes after contamination and should be gently rubbed into the skin for at least five minutes. (The use of bleach ointment or cream is not recommended for animals since it is more irritant to animals' skin than bleach paste.) In the case of lewisite splashes, in addition to the above first-aid treatment, the application of warm water, preferably containing an alkali such as washing soda, is effective provided treatment is applied immediately, but even so some redness of the skin is likely to result.

Lewisite blisters should be punctured to allow the fluid content to escape, but mustard gas blisters should not be punctured. The reason for this differentiation is because lewisite is an arsenical poison. Subsequent treatment is the same as that for ordinary burns and scolds. Extensive injury or contamination by either of the blister gases necessitates skilled attention.

PERSONAL PROTECTION AGAINST THE BLISTER GASES.—The respirator gives protection only to the covered parts of the face and respiratory system. It is usual to provide A.R.P. workers with protective suits if their duties are likely to bring them in contact with heavy contamination in either liquid or vapour form. Such protective clothing consists of rubber wellington boots and oilskins. In heavy concentration of mustard gas oilskins provide efficient protection for three or four hours only, after which period the clothing must be decontaminated by boiling in water. Oilskins which have been boiled become less resistant to liquid gases and are only serviceable for use on three or four occasions—each time they are boiled the resistance to blister gas becomes less and the clothing should be worn for increasingly shorter periods.

Effects of the Blister Gases on Animals

Mustard Gas, either as liquid or vapour, attacks any part of an animal with which it comes in contact. The horn of the hooves of the larger animals is, however, sufficiently resistant to afford protection. In these animals, the cushions of the heels, being covered only by fine skin and being very near the ground, render the back of the foot the most likely part to be attacked by liquid blister gases. Tissue injured by mustard gas heals slowly and is very prone to become septic. Fortunately the greater part of the body surface is protected by the animal's natural coat which serves as a first line of defence against liquid gas. In the case of the horse and other animals, liquid gas tends to pass down the hairs and collect as small droplets, and this point must be borne in mind when handling contaminated animals or carcasses.

In the larger animals the skin is comparatively resistant to mustard gas vapour, but injury may be sustained if the concentration is high and/or the exposure prolonged, particularly between the legs where the skin is fine. The

risk is greater when the animals are sweating or wet. Injury to the skin by vapour may be caused when the liquid gas falls upon the harness or clothing. The injuries resemble those caused by direct liquid contamination except that they are milder in character and healing takes place in a shorter time.

Unlike man, the hair-covered skin of animals does not blister when contaminated with mustard gas. A swelling marks the area affected, but this subsides, leaving a hard, wrinkled area of dead skin which finally separates and leaves an ulcerated area. With light contamination the skin may be injured superficially only and new hair may grow when the dead skin has flaked off. Heavier contaminations cause deep-seated ulceration and sloughing. In favourable cases healing will take eight to ten weeks.

An animal's clean, long coat affords good protection against mustard gas vapour. If the coat is long but dirty with an accumulation of grease and dandruff the lesions may be more severe than those resulting from the gas contamination of a clean clipped animal. When exposed to mustard vapour the eyelids swell, tears run down the face and later there is a thick discharge. The smallest drop of liquid gas in the eye causes serious injury. Within ten minutes there is a copious flow of tears and the animal rubs the affected eye. In the acute stage the eyelids are closed and temporary blindness results. This stage passes off in the second week, but complete resolution may take months. Heavy contamination may lead to permanent blindness.

Inhalation of the vapour causes ulceration of the lining membrane of the nose and windpipe. A thick, nasal discharge develops and this may be streaked with blood. A sudden rise in temperature may indicate the onset of complications, e.g., pneumonia. Animals moving over contaminated ground may receive injuries to the limbs according to the nature of the ground surface. On grassland, the front and sides of the legs are chiefly affected, but where the "going" is heavy, as on plough or muddy land, the fine skin of the heel and the back of the pastern and that between the claws or toes of cattle, sheep and pigs will be affected. The hocks may also be injured from splashing. The injuries are particularly prone to secondary infection and may cause intense lameness, inability to seek food at pasture and consequent loss of condition.

Grazing over contaminated pastures or the eating of contaminated fodder causes ulceration of the mouth, stomach and intestines. In mild cases the animal salivates freely and is unwilling to feed when the ulceration is established. In severe cases there is abdominal pain (colic) and diarrhœa, the excreta being offensive and bloodstained.

Lewisite acts in a similar manner to mustard gas except that it is more irritant. It may cause general poisoning owing to the absorption of arsenic.

PREVENTIVE MEASURES.—No satisfactory animal gas mask has yet been produced for general use. Apart from the difficulty of training animals to tolerate such devices, there are other objections. For example, when a dog

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is fitted with a mask he loses his sense of smell and with it his sense of direction. Excessive salivation is also an objectionable feature. Also the heat-regulating mechanism is seriously affected. The respiratory volume of the larger animals necessitates a bulky apparatus difficult to harness to the head. Gas-proof kennels fitted with an appliance which enables the occupant to breathe air drawn through a gas filter is probably the most efficient appliance obtainable.

Farm Animals.—The natural resistance of the skin of horses and cattle, and the protective value of the fleece of sheep, would discount to some extent the effects of air-sprayed mustard gas. Animals should be driven from contaminated pasture. Improvised foot baths of bleach paste are useful if employed *early* after exposure. If clean standings are not available animals should be penned or folded on straw.

The thick, long coat of the horse affords a good measure of protection for the skin against the effects of blister gases. When clipping of the coat is necessary in the case of working horses this should be restricted to the belly.

Rugs, blankets, eye-fringes and other clothing will give some protection against air-sprayed mustard gas provided they are removed as early as the situation permits, e.g., on gaining the shelter of stables, etc. If there has only been brief contact they will be safe after forty-eight hours' weathering. Good protection to the legs can be secured by bandages, provided they are removed early after contamination. Bandages treated with boiled raw linseed or other vegetable oils and " Dunlop " boots give the best protection.

Anti-gas ointment smeared on the legs before exposure, particular attention being paid to the fine skin of the heels, gives excellent protection. It is an advantage if the ointment is worked over the legs by hand rubbing. To avoid bleach irritation the ointment should be removed as opportunity offers.

Anti-gas ointments with a mineral jelly base are recommended as those with a vanishing cream base are too irritant.

Small Animals and Birds.—Persons having household pets in their possession during air raids should take them with them into a gas-proof room or shelter. One large dog or two small dogs or cats should be considered equal to one person in calculating the air space available in gas-proof shelters or rooms. The protection of poultry, cage-birds, and other small pets in air raids presents a difficult problem.

All birds, and especially small cage-birds such as canaries, are very susceptible to poison gas, and exposure to even relatively weak concentrations is likely to prove fatal.

The only solution in the case of poultry and other small pets is to keep them either in a gas-proof room or in a gas-proof house.

Gas-proofing of Buildings, etc.—A not inconsiderable measure of protection against the effects of " gas " can be afforded by *gas-proofing* buildings and no difficulty need be anticipated in the case of well-constructed buildings.

Blankets or even layers of sacking, etc., can be used for the inside of doorways and windows, which can be made to close tightly by means of a felt strip. Small cracks and holes in doors can be filled with putty, mud, etc., or stout brown paper may be stuck over them. For metal-framed windows gummed paper strip is recommended.

Special arrangements must be made for the various types of ventilators, and drainage and other exits must be closed—wet hay, wet newspaper pulp, cotton waste or rags are useful for this purpose. Many farm buildings are constructed of weather boarding, open timbers, corrugated iron, or are in such a state of disrepair that they cannot be readily rendered gas-proof. Rick sheets, tarpaulins, and similar materials may be used to help render such buildings gas-proof. Even some limited protection is better than none at all and may materially assist in preventing the concentration of gas reaching dangerous proportions. Buildings may be overcrowded in an emergency for animals can withstand much better than human beings the vitiated atmosphere brought about by lack of ventilation and can therefore be confined in such buildings for a time long enough to ensure the dispersal of non-persistent gases.

In the case of contamination by aerial spray, the situation with regard to the buildings does not differ materially from that existing elsewhere. The more open situation of farm buildings generally will, however, be in their favour with regard to the beneficial effects of weathering.

The roosts of poultry houses should be made as gas-proof as possible by blocking up any holes in the woodwork and the doors should be made to fit as tightly as possible. Sacks and blankets should be nailed over the top of the doors and folded and tied up above the doors in such a way that they can be released easily and allowed to fall over the door in case of an emergency. If time will allow they should be saturated in water.

Where holes are provided for ventilation, similar arrangements must be made to render the ventilation gas-proof. Where it is found impracticable to arrange blankets or sacks in the manner suggested above, wet sacks, old rags or even wet hay or straw may be pushed into the ventilation holes.

In the case of cage-birds and other small pets kept in cages, such as mice, some degree of protection can be obtained by folding a piece of material, e.g., flannel, etc., round the outside of the cages. The material should first be soaked in water.

The Protection of Water and Food Supplies

WATER SUPPLIES.—No great danger from poison gas contamination with regard to water supplies is to be expected. True gases and vapour do pollute water. Liquid blister gases sink in water, but may leave an oily film on the surface which will be particularly noticeable on still water. There is some danger of arsenical poisoning from water contaminated by lewisite. Watering from shallow ponds should be avoided, but if the use of such water is abso-

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lutely necessary then the supply should be scooped from the surface. No danger need be anticipated from running water (streams). Contaminated troughs should be drained and treated with bleach paste. The water authorities will, no doubt, deal with any widespread danger to public supplies, and so far as private supplies are concerned, such as tanks, etc., the provision of adequate covering should suffice. However, should the water taste or smell peculiar, expert advice should be sought before its use is sanctioned.

FOODSTUFFS.—In view of the fact that poison gases are highly penetrative and that foodstuffs are often very absorbent, general protective measures are indicated. Any material which soaks up water or oil, or absorbs odour, will absorb poison gas. If this knowledge is combined with a fair conception of the protective powers of various types of packings the protection of foodstuffs becomes largely a matter of common sense.

Non-persistent gases (e.g., chlorine, phosgene), in ordinary concentrations, only affect foods temporarily and the effect soon passes off, although in higher concentrations and after a more prolonged exposure they will produce deterioration. Palatability is, of course, affected, but the food itself is not necessarily made unwholesome.

The nasal irritant gases (D.A., D.M., D.C.) **and arsine.** — With the arsenicals which are released in fine particles, and which are readily dispersed, the effect on food would be slight, but there might be a risk of arsenical contamination, so that the decision as to whether the foodstuffs are fit for consumption rests upon expert examination. The slightest suspicion that arsenical gas has affected the food should at once indicate that expert advice is to be sought before deciding on its fitness for consumption.

Tear gases only affect palatability and airing would be of value, unless the foodstuffs are highly contaminated.

Blister gases of the **non-arsenical** type (e.g., mustard). — Slight contamination may only affect palatability, but weathering for 48 hours and cooking should render foodstuffs safe. Fatty foods, however, should always be regarded with suspicion after contamination in view of the readiness with which blister gases penetrate oily substances. When food is contaminated with blister gases of the **arsenical type** (e.g., lewisite) it should be looked upon with the greatest suspicion, owing to the danger of arsenical poisoning, and expert advice should be sought.

TYPES OF PROTECTIVE PACKINGS.—Complete protection can be given against all forms of gas by airtight bottles, glass or earthenware jars or sealed tins. Waxed cartons and oilskins give good protection against vapour and liquid. Greaseproof paper gives slight protection against liquid contamination, but is effective against vapour. Cardboard and ordinary paper gives only a limited protection against vapour and no protection against liquid. Ordinary sacks and textile coverings have no protective value when filled with food.

Where foodstuffs are to be kept in bulk, poorly protected against gas con-

tamination, measures should be taken to render the store place itself gas-proof as far as possible. War gases being heavy, the upper floors of buildings are indicated as store places although it must be remembered that the risk of incendiary bombs is greater in such situations.

Open sheds afford little or no protection, and foodstuffs kept in them should be covered with tarpaulins. The latter vary in efficiency. Those of the impervious oil-dressed type are best. It is best to have the tarpaulin suspended on a framework so that it is not in actual contact with the food.

Even pervious material, however, will give some protection to food provided an air space is interposed between the covering and the food. Pervious coverings are easily decontaminated by simply airing for a few days or, better still, by boiling in water.

Growing Crops.—The effect is only likely to be transient—the greatest damage may be caused by spray from low-flying aircraft.

Arsenical gases, such as lewisite and arsine, would be a more serious problem, for arsenic is a powerful plant poison. Weathering will, in time, remove the danger, but the length of time would depend on the amount of concentration and the type of gas used. However, should a gas bomb explode amongst growing crops or should gas spray be used the police or local authorities should be informed at once.

The danger of walking over blister gas contaminated fields should not be overlooked. Should this, however, be necessary, e.g., to attend injured stock, full protective clothing must be worn.

It is not advisable to turn animals out to graze on pasture land which has been contaminated by the spray method until at least 48 hours after the contamination has occurred.

Growing and root crops will require special consideration according to the stage of their development, the nature of contamination, and the state of the weather. The advice of the local gas officer should be obtained with respect to such crops. If there is still some weeks to go before the crop is gathered in, and the spray is of a light order, the corn or roots may be quite safe for consumption, but one should obtain expert advice before utilising it for food.

If grassland or crops are subjected to heavy concentrations of non-persistent gases they may be damaged or, in some cases, destroyed. The effect of gas in such circumstances will generally be observed by the bleached appearance of the grass or crop. No immediate action is, however, called for, and if the crop recovers and grows there should be no subsequent danger.

With regard to farmland which has undergone spray contamination, one can with reasonable safety rely on the decontaminating effects of weathering. In mild or warm weather there will generally be little danger remaining after three days. In cold weather the danger may persist much longer and in such circumstances precautions should be observed as indicated in the general instructions issued in this connection.

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Stacks.—None of the poison gases likely to be used would seriously affect stacks of hay, etc., although it must again be emphasised that arsenical gas contamination should be looked upon with more suspicion. In any case, removal of the outside of the stack should be a reasonable safeguard.

Fodder.—Fodder exposed to the true gases, arsenical smokes or the vapour of blister gas will not remain dangerous, but will lose its palatability. Sacks and bales should be opened and the contents aired until decontaminated. Animals usually refuse to eat gas-contaminated fodder. Forage, etc., which has been contaminated or suspected of being contaminated with liquid gas should not be handled or used until it has been inspected by the local gas officer or has been allowed to weather for at least 48 hours. The longer it can be left the greater the safety.

Meat Supplies.—Emergency slaughter may be necessary. Animals must be thoroughly bled after slaughter and should be skinned, eviscerated and hung up with least possible delay. Failure to bleed properly and promptly to eviscerate the animals will render the meat unsuitable for human food. It has not been definitely determined as yet whether animals which have been exposed to gas will be fit for human consumption. This will depend on the circumstances. In the case of an animal which has been affected by the inhalation of a choking gas the carcass will be probably fit for consumption. It is essential that such animals should be slaughtered in the early stages before fever sets in.

When an animal has been contaminated with mustard gas some time elapses before symptoms arise. If the contamination is widespread it should be slaughtered immediately. The flesh would be fit for consumption after the contaminated parts and underlying tissues have been cut away. If it is known that an animal has swallowed food contaminated with the blister gases slaughter should be carried out as soon as possible in order to avoid contamination of the flesh. In the case of widespread contamination by lewisite the carcass is unfit for consumption and must be rejected.

When gas-contaminated animals are to be slaughtered it may be possible to decontaminate sufficiently before slaughter. If this is not possible or advisable the decontamination may be carried out immediately after slaughter and prior to dressing. If the carcass of an animal suffering from gas poisoning is passed as fit for human food the offal should, in such cases, be rejected.

Dressed Meat.—Meat contaminated with phosgene may be slightly discoloured, but the normal appearance will return on exposure to fresh air, and then it may be passed as fit for food. Dressed carcasses which have been exposed to blister gases are not likely to be fit for human food, for these gases are readily soluble in animal fats. In the case of slight vapour contamination it may be possible to remove the superficial layers of the meat, but this decision must rest with the local meat inspector who will, if necessary, submit the meat to analytical examination—especially in the case of contamination with lewisite.

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Treatment of Casualties

It should be emphasised that in all attacks in which the gas warning has been given steps must be taken to ascertain whether gas contamination has occurred before treatment of any kind is attempted. If this is confirmed or even suspected the patient should be decontaminated, and every precaution taken to protect the person attending the animal.

HIGH EXPLOSIVE BOMB INJURIES.—These may be due to fragments of bomb casing, to stones or other material flung by the explosion, and to falling masonry and debris. Under this heading must also be included wounds due to falling shrapnel and splinters from anti-aircraft fire. Many wounds caused by bomb casing will be of the severe lacerated type, others may be deep and penetrating. Vital organs may be penetrated, parts of the body may be shot away, bones may be fractured and foreign bodies may be implanted in the tissues. Wounds from anti-aircraft fire will be less severe. Falling debris may cause crushing, bone fractures, contusions and wounds of all types. Frenzied struggling by the animal may aggravate these injuries. The animal should be suitably restrained and then carefully examined to note the extent and nature of the injury.

All projectile wounds will require skilled surgical treatment. First-aid treatment will comprise the cleansing and protection of the wound and the arrest of severe hæmorrhage. It is inadvisable to probe the wound when rendering first aid, for infection may be introduced into an otherwise clean wound. Cover the wound with lint or cotton wool soaked in water or an antiseptic and then clip the hair away from the edges. The wound and the surrounding parts should then be thoroughly cleansed with running water or soap and water. The area should then be swabbed with acriflavine solution, eusol or similar antiseptic, the wound plugged with gauze or muslin soaked in the antiseptic and protected with a bandage or adhesive plaster. If antiseptic solutions are not available a handful of table salt to half a pailful of water will serve as a temporary dressing solution.

Larger wounds should be treated in like manner. Extensive bruises can be relieved by hot or cold fomentations with antiseptic solutions or salt water.

EXTENSIVE BLEEDING from wounds should be controlled. Packs or plugs used to stop bleeding should be left in position for at least two days. In those parts of the body which it is impossible to bandage dressings may be kept in position by strips of adhesive tape.

Attendants and owners should be instructed that the arrest of hæmorrhage by tight bandaging or tourniquet is only a temporary measure and must be replaced by proper surgical methods *as soon as possible*.

An injection of anti-tetanic serum is a wise precaution.

No strict rules can be observed in these cases; treatment must depend on the nature of the injury and the circumstances at the time.

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SHOCK.—Animals may suffer severely from shock. They should be kept quiet and warmly clothed; brandy, whisky, or sal volatile in small doses and well watered will afford temporary relief.

BURNS AND SCALDS.—Such injuries, produced by fires or by boiling water or steam from damaged boilers or hot water pipes, may vary greatly in their degree of severity. In the case of very serious or very extensive burns or scalds one should first consider whether the injuries are such that treatment is practicable. In many cases the animal should be destroyed as soon as possible. Tannic acid is invaluable in the treatment of burns. Tannic acid jelly, a solution of tannic acid in water, or strong tea that has been allowed to stand are excellent forms in which it may be used. These are best applied either by lightly wiping them on the parts with cotton wool or, even better, when a liquid is used, by means of a sprayer. Afterwards the part should be protected with lint or cotton wool or dusted over with a soothing powder, such as toilet powder, talcum powder, flour or starch, or calamine powder. Any of these powders may be made into a paste with the white of an egg. Other useful dressings are cod liver oil, acriflavine emulsion, solution of picric acid, a strong solution of epsom salts in water, carron oil, olive oil, lime water and milk, treacle or medicinal liquid paraffin. Tannic acid solution applied with a garden spray is useful in injuries to a cow's udder. A sedative may be necessary in order to relieve pain or quieten the animal.

POISON GASES.—It is possible that blister gases may be employed together with high explosive and incendiary bombs, thus resulting in wounds, fractures, burns and other injuries being complicated by "gas." The effects of poison gas depend upon concentration and the time of exposure.

MUSTARD GAS.—When contaminated animals have to be treated the attendants must, for their own safety, wear respirators and protective clothing. In the absence of gloves, hands and all exposed skin should be thoroughly smeared with anti-gas ointment.

The success of first-aid treatment depends upon the speed with which it is applied. Unless it is employed within *ten minutes* after contamination by the liquid, injury will follow. The longer the treatment is delayed the worse will be the injury, but some mitigation may be expected even when the delay amounts to hours.

In the case of transport animals the attendants should:

(a) Remove saddlery and harness, which should be regarded as contaminated.

(b) Secure the tail by holding or tying. Before doing so, dust with bleaching powder, bleach and sand mixture or soak with bleach solution, eusol, or a solution of sodium hypochlorite (Milton, chloros, voxsan, etc.).

The animal should then be treated as follows:

DECONTAMINATION

(1) Where the contamination is widespread and cannot be accurately defined:

Scrub the animal all over vigorously with soap and water. Warm water, soda and soft soap will be most convenient. Use plenty of water and continue washing for twenty minutes; or brush into the skin a paste made of equal parts of bleaching powder and water, care being taken to avoid the eyes, nostrils and lips. Bleach paste is irritant and should not be left on the skin longer than five minutes, but with hairy animals it may be left on for longer periods—it is removed by flushing with water; or soak with eusol or a solution of sodium hypochlorite; or hose down with water for half an hour. (NOTE: Bleach paste should preferably be freshly prepared for use as it tends to solidify after some hours and has to be further diluted with water to render it fit for use. The weaker solution is less irritant to the skin and so may be applied for a slightly longer period. If it is necessary to store bleach paste for short periods it should be kept in wooden buckets. Pastes made with a fatty basis are unsuitable for application to animals because of the difficulty of their subsequent removal.)

(2) Where the contamination is local and clearly defined:

If the drops are visible they should be removed by moist tow, cotton wool, cotton waste or rags, care being taken not to spread the contamination to the surroundings parts. The cotton wool or waste is promptly burned or buried. Affected areas are then treated with bleach paste and washing, or by the application of anti-gas ointment. Anti-gas ointment is itself irritant and must be removed as soon as opportunity offers. The bulk should be scraped out of the coat and the remainder removed by petrol. It is on account of this difficulty in removing anti-gas ointment from the coat that the use of watery bleach paste is a more convenient method for animals. Swabbing well with eusol or a solution of sodium hypochlorite is also a method of decontamination. In certain circumstances it may be convenient to swab off the gas by solvents such as paraffin or petrol. It must be realised that the gas is not destroyed by this method but is simply diluted. Care should be taken, therefore, not to spread the contamination to the surrounding parts when swabbing with solvents.

Eyes should be treated in the following manner:

Wash out freely with plenty of plain warm water or with a solution of bicarbonate of soda or common salt (one teaspoonful to a pint of water). Continue to irrigate for ten minutes. Then put a little cod liver, castor or medicinal paraffin oil into the eye.

When the **lungs** are affected rest and good nursing are essential. The animal should have plenty of clean drinking water and an ample supply of fresh air. Rugs and bandages should be used to maintain body warmth.

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If the coat of the animal is dirty with an accumulation of grease and dandruff the lesions may be more severe than those resulting from contamination of a clipped animal. A clean, long coat affords good protection against mustard gas vapour.

LEWISITE.—First-aid treatment consists of washing well with hot water, soda or soap, or water alone, but it should be carried out within 20 minutes after contamination. Eyes should be treated as advised for mustard gas.

CHOKING GASES.—(a) Large animals. The first essential is to remove the animal from the dangerous atmosphere. To prevent the inhalation of more gas during this stage, nosebags lined with damp hay should be put on, or two nosebags telescoped with damp hay or chaff between them may be used. Harness and saddlery should be removed from horses, which should be moved slowly, with frequent rests, to a place of safety, i.e., upwind of the gassed area or to high ground. Excessive movement or the lightest work will aggravate the condition of the animal and may lead to death. If ambulance transport is available it should be used. Keep the animals in the open air, putting on rugs and bandages for warmth. *What appear to be slight cases should be treated as serious cases because of the possibility of graver symptoms developing later.* Convalescence is slow. Horses should be brought gradually and carefully back into work after recovery.

(b) *Small animals.* Small animals should be removed at once from the dangerous atmosphere and in so doing protection should be provided by swathing the head or the entire animal in a blanket. They should be kept confined, but *allowed plenty of fresh air,* warm clothing and good nursing.

Animals which have been subjected to any of the choking gases should be placed under veterinary care as early as practicable.

THE STUDY OF COCCIDIOSIS (EIMERIA CAVIÆ [Sheather 1924]) IN THE GUINEA PIG

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PART II (Continued)

The Merozoites

The merozoites of *E. caviæ* have the sickle-shaped form typical of coccidian merozoites in general and, when they are alive in scrapings of the mucosa put up in normal saline or Ringer's solution, in which they live for some minutes at laboratory temperatures, they execute the familiar gliding

and sudden bending movements that are characteristic of the merozoites of all species of coccidia. The nucleus can sometimes be seen as a clear area floating at about the middle of the merozoite. In this is a light shadow, which represents the karyosome and the extrakaryosomatic area of the nucleus. In these preparations merozoites closely resemble sporozoites that have been liberated from the oocysts by the action of digestive juices.

Their appearance in fixed and stained preparations is shown in Fig. 12. These figures show that the nucleus, like the resting nucleus of the other phases, has a single karyosome situated at one pole of a pear-shaped extrakaryosomatic area, which itself shows the different appearances depicted in the figures, according to the degree of differentiation of the stain, differences which have been already explained. More than one karyosome may be present in the nucleus of the merozoite of some species of coccidia, but more than one has never been seen by the writer in the nucleus of the merozoites of *E. caviæ*. Nor is there ever more than one nucleus in each merozoite, so that the question whether division of the nucleus may occur before the merozoites are liberated from the bundle of them formed by splitting of the schizont does not arise. Schellack and Reichenow (1913) found that the nucleus of the merozoites of *Barrouxia schneideri* may contain more than one karyosome even before the merozoite is liberated and they thought that this indicated that the schizont nucleus, which is the nucleus of the merozoite, may thus begin to multiply very early before the merozoite has actually become a schizont.

The writer has not been able to determine how many generations of merozoites are produced by *E. caviæ*; nor has it been possible to determine exactly how the schizont splits to form the merozoites. In sections the edges of fully-grown schizonts often show irregularities which could be interpreted as buds of cytoplasm, suggesting that the merozoites arise, as those of some other species do, by budding, but it is likely that these irregularities are due to the action of the fixatives. If the experiments that are being done by the writer on the artificial cultivation of *E. caviæ* succeed, it will be possible to determine this point more accurately.

The granules which are commonly seen in the cytoplasm of coccidian merozoites, granules which usually stain deeply with hæmatoxylin and are usually regarded as being granules of food material (volutin, etc.), have not been seen by the writer in the merozoites of *E. caviæ*.

It is never possible, in guinea pigs which have been artificially fed with oocysts and killed at definite intervals afterwards, to find only the stages of the sexual cycle; stages of the asexual cycle are always present as well. This indicates that the sexual cycle does not begin suddenly and involve all the merozoites present, but at first affects only a proportion of them, so that the others continue to give rise to more schizonts, the asexual and sexual cycles going on for a while side by side. This is true of most species of coccidia. The duration of the asexual cycle is not determined entirely by factors operat-

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ing within the parasite, but must be affected by the host's resistance to it, which influences the nutrition of each phase of the parasite. It must also happen that some of the sporozoites emerging from any particular dose of oocysts will not develop as rapidly as the others, either because they belong to a less vigorous strain or because they happen to encounter localised less favourable conditions inside the host. They will therefore complete their quota of sexual generations later, when the others have already given rise to sexual phases. Schizogony cannot therefore be expected to cease entirely before the sexual cycle begins. Other factors may also be operating to produce this result, of which we shall remain ignorant until we can cultivate a species of coccidia and study questions like these by experimental methods. The writer hopes in future to be able to describe a method of doing this.

This question of the duration of schizogony is, indeed, inseparably bound up with a number of other questions raised by the study of various species of coccidia, such as the number of generations of merozoites produced, the variations in the number and size of the schizonts and the merozoites which they form, the precocious sexual differentiation of the schizonts which occurs in some species and similar problems. The answer to all these questions is probably to be found in the study of the interaction of variations in the resistance of the host with variations in the vigour of different strains of the parasite. These two controlling factors must also determine the general question of the adaptation of each species of coccidia to its host and consequently its pathogenicity. We cannot, until we can cultivate a coccidian species in a controlled artificial medium and can also control its host, prove that strains of it of varying vigour or virulence occur, nor can we determine exactly what factors govern the variations in the size and number of the different phases of the life history which all species of coccidia show; nor can we determine, without experimental control, exactly the factors on which the resistance of the host depends. We are, in fact, so long as we have no experimental means of study, little better off than the earlier workers who had to appeal to the exhaustion of a "biological impulse" to schizogony, which eventually waned, so that the asexual phases ceased and the sexual phase began. But such knowledge as we have of resistance to coccidian infestations and of the variable sizes, numbers and pathogenicity of the different phases of the parasites can be reduced to the general statement that the nutrition of the parasite governs all the phenomena observed.

In resistant guinea pigs, for example, the schizonts of *E. caviæ* will produce fewer and smaller merozoites; in non-resistant ones they will produce larger and more numerous merozoites; and there is no reason to suppose that the smaller or larger ones belong to different species. For the same general reasons a vigorous strain of *E. caviæ* will rapidly overrun the colon of a non-resistant guinea pig, producing large and numerous merozoites at first; but it will thus quickly exhaust its host and the cells available, impoverishing the

healthy cells that remain, so that its later generations of schizonts and merozoites will be smaller and fewer, until its schizogony ceases; and, if the host survives and no further infestation occurs, the infestation will naturally cure itself by the conversion of all the parasites into oocysts which will be passed out of the host. On the other hand, if a relatively feeble strain of *E. caviæ* infests a resistant host, we shall expect to find that it produces fewer and smaller schizonts, which will give rise to fewer and smaller merozoites and that schizogony will not produce so many generations of merozoites. In some hosts we shall expect to find also that the resistance of some localised areas of their tissues will be stronger than that of other areas, so that the numbers and sizes of schizonts and merozoites will vary even within a particular strain, larger schizonts which split into larger and more numerous merozoites being produced in areas of the tissues of weaker resistance and smaller schizonts, giving rise to smaller and fewer merozoites, being produced in areas resisting more strongly. It is no doubt for these reasons that the late schizonts of *E. præcox* in chickens produce, as Tyzzer, Theiler and Jones (1932) found, merozoites of different sizes, varying in number from 8 to 48, and that *E. necatrix* in chickens, after producing several generations of merozoites, develops either small schizonts which produce small merozoites or very small schizonts which produce minute merozoites and that these late merozoites do not cause, in the cells which they infest, the reaction produced by the earlier generations of larger merozoites. Evidently they are the products of exhausted schizogony.

Similar reasons are probably responsible for the fact, observed by Schellack and Reichenow (1913), that the number of merozoites produced by *Barrouxia schneideri* in the intestine of *Lithobius forficatus* varied from 6 to 30, but never in any one infestation encompassed the whole of that range and that fewer and smaller merozoites were produced when schizogony was nearing its end. They thought that variations of this kind, like the variations in the sizes of other phases of the life history, depended on the nutrition of the parasite.

The same general explanation can probably be applied to Schaudinn's (1900) statement that the smaller merozoites of *Eimeria schubergi* were produced because the nucleus of the schizont began to divide at a very early stage in its growth. It is well known that the rates of division of the different nuclei within the same schizont may vary considerably, as they do in the schizonts of *E. caviæ*. Nutritional factors probably also govern the precocious sexual differentiation of the asexual phases which some species of coccidia show. Reichenow (1910), for example, showed that *Hæmogregarina stepanowi* produces larger schizonts, measuring 20 x 13μ, which produce 12-24 merozoites and smaller schizonts, measuring only 16 x 6μ, which produce only six (more rarely, four or five) merozoites, these latter merozoites always becoming gametocytes, so that the size of the smaller asexual phases is

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associated with a precocious sexual differentiation. Robertson (1910) showed that *Hæmogregarina nicoriæ* exhibits a similar differentiation. In both these species microschizonts produce fewer and smaller merozoites which always become gametocytes, while the macroschizonts produce more numerous and bigger merozoites, which either grow into macroschizonts again or into microschizonts.

When we remember all these facts, it is not surprising that the writer found that the size of the merozoites of *E. caviæ* varies from 6-16μ and that it is difficult to state precisely how many merozoites are produced by the schizonts. In transverse sections of the bundles of them 20-24 or more can often be counted, but many bundles contain only 12 or fewer. Sheather (1924) found a maximum of 32. Henry (1932) is content to quote this maximum.

An appearance shown by the transverse section of these bundles of merozoites may be mentioned here, because it may confuse the investigator. Transverse sections of these bundles may give the appearance of a number of spherical bodies grouped together in what appears to be the remains of a schizont that has split up into spherical pieces each with a nucleus. Simond (1897) and Wasielewski (1904) both figure these. The correct interpretation of them is, not as Simond and others thought, that the schizont splits up into spheres and that these then elongate to become the merozoites, but that, as Wasielewski points out, the spheres are transverse sections of processes of the protoplasm of the schizont or of fully formed merozoites. When sections pass through the nuclei of the merozoites the appearance of a sphere with a central grain is obtained. Even when the section does not go through the nucleus, the merozoites may, especially in preparations overstained with iron hæmatoxylin, take the stain in such a way that they appear to be spheres with a central grain-like nucleus. The diameter of these apparent spheres is the same as that of the merozoites, namely, 1-1.5μ. Some collections of these apparent spheres seem to be enclosed in a definite membrane; but others do not, nor is such a membrane enclosing the bundles of merozoites visible in longitudinal sections of the bundles. The writer is unable to state definitely that a membrane encloses the merozoite bundles, so that it seems preferable not to use the term " merozoite cyst."

The merozoites are not confined to the epithelial cells which they infest. When the infestation is heavy, they may be found in masses in the lumina of the crypts of Lieberkühn, and in the lumen of the colon itself. In these respects they differ from the merozoites of some other species which are not found in the intestine of their hosts, but live all their lives in the cells which they infest or in the tissues through which they migrate in order to reach these cells.

Abnormal or degenerate merozoites, such as those recorded by Schellack and Reichenow (1913) and by Tyzzer (1929) have not been seen, but no doubt

they occur, especially in resistant guinea pigs and they may puzzle the investigator.

The Schizont

The very young schizont, which is formed by the shortening and broadening of the sporozoites or the merozoite, is shown in Figs. 1 and 13. It is usually oval in transverse section and its cytoplasm is free from inclusions. The appearance of its nucleus, like the appearance of the nucleus of the other phases, varies according to the degree of differentiation of the stain. The divisions of the nucleus, like those of the nucleus of the schizont of many other species, begin very early in the development of the schizont, which does not remain uni-nucleate until the schizont has reached a certain size, as the schizonts of some species of coccidia are said to do. These amitotic divisions of the nucleus have already been described. The division of the karyosome is often, but not always, unequal and the nuclear divisions, like those of the schizont nucleus of *Schenococcidium intermedium*, described by Léger and Duboscq (1910) and other species of coccidia, are not synchronous, some nuclei appearing to divide faster than others. For both these reasons schizonts are often found which contain either an odd or an even number of nuclei which may be unequal in size (Figs. 9 and 10). It has not been possible to decide whether the nuclei which divide faster produce healthier merozoites than those which divide more slowly. Whether the divisions later become more nearly synchronous it is difficult to determine, but certainly the nuclei appear to be more nearly equal in size when they have reached the number of 14 or more (Fig. 11). By then they have begun to take up positions near the surface of the schizont, rather than its interior.

At first, while its nucleus is single, the young schizont is indistinguishable from either the young female or the young male gametocyte. All these phases have clear cytoplasm, free from granules or other inclusions, and their size does not help us to differentiate between them, because they have not completed their growth; and in any event, the normal size variations of all phases of the parasites is considerable. Once the nucleus has divided, however, it is possible to say that the development of either a schizont or a male gametocyte has begun, because the nucleus of the female gametocyte does not divide at any stage in its development. It always remains single and is always somewhere in the interior of the cell, never at the surface, as the nuclei of the schizont or male gametocyte may be. Further, the nucleus of the female gametocyte stains rather more deeply and resists differentiation, so that the halfmoon-shaped extrakaryosomatic area near the karyosome, seen so often in the nuclei of young schizonts and young male gametocytes, is less often seen in the nucleus of female gametocytes. It is hardly possible to determine accurately whether the nucleus of the young female gametocyte is larger in

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proportion to the amount of cytoplasm present than that of the uni-nucleate schizont, but there appears to be little evidence that this is so. The young female gametocyte may also be distinguished from the young schizont by the fact that, very early in its development, its cytoplasm begins to show minute, spherical granules, some of which are destined to form the walls of the oocyst and some are no doubt reserve food material (Fig. 26). Granules of this kind are never seen in the schizont or male gametocyte at any stage in their development.

From the young male gametocyte the young schizont cannot be distinguished at all. Like the corresponding stages of other species of coccidia, these two remain similar in every respect until the number of nuclei produced by the male gametocyte has exceeded the maximum number ever produced by a schizont. It is difficult to state precisely either the normal or the maximum number of nuclei produced by a schizont, because the development of all stages depends on the vigour of the strain of parasite being studied, on the resistance of the particular host and on any other factors which influence the parasite's development. No record exists, however, of a higher number of merozoites of *E. caviæ* than the 32 which Sheather (1924) records, so that it can be said that, when more than 32 nuclei are present, these are the nuclei of a male gametocyte and not of a schizont.

The normal full-grown schizont, which therefore contains 16 or 32 nuclei or so, is usually oval in shape, with its nuclei at or near the surface, so that its appearance is characteristic. In badly fixed preparations schizonts with 14 or more nuclei seemed to have a tendency to split up into areas of cytoplasm round each nucleus. Such schizonts may have been ready to form the merozoites. When the merozoites are fully formed the familiar "corps en barillet" figure, like an orange with its sections exposed, is produced (Fig. 12). The residual body, namely, the protoplasm left over after the merozoites have been formed, is not always present. It is absent, for example, from the bundle shown in Fig. 12, but is well marked in those shown in Figs. 18 and 19. The reason for this is obscure. Schellack and Reichenow (1913) only rarely found a residual body in bundles of the merozoites of *Barrouxia schneideri* and never found it in wet preparations. They think it is produced by external influences which perhaps operate when preparations are being made. The fact that the residual bodies shown in Figs. 18 and 19 are drawn from sections fixed with formol-saline, while the bundle shown in Fig. 12, which has no residual body, was drawn from a section fixed with Helly's mixture, a much better fixative, would seem to support this view. There is no doubt that in the past too much significance has been attached to the residual body and, when coccidia can be artificially cultivated, we may find that it is often, as Schellack and Reichenow suggest, an artefact or that it may be an expression of deficient vigour in the schizont, appearing perhaps only in bundles formed at the end of the schizogonous cycle or in strains of the parasite that are less

vigorous than others; the resistance of the host may likewise so affect the parasite that it fails to use all its cytoplasm for the formation of merozoites, so that a residual body is left.

No attempt has been made in this investigation to answer the important question asked by Andrews (1930), whether there is, in coccidian infestations in general, a quantitative relation between the numbers of schizonts produced and the number of oocysts passed in the fæces. He points out that this question is important because the degree of infestation in any animal is usually judged by the number of oocysts being passed out of it, while the damage done to it is done, not by the oocysts, but by the asexual schizonts and the merozoites they produce. No doubt important questions like this one cannot be satisfactorily answered until we can cultivate a coccidian species artificially and subject its normal cycle to experimental control.

The Male Gametocyte and the Formation of the Male Gametes

The male gametocyte, like that of other coccidia, produces many more nuclei than the maximum number produced by the schizont. Schellack and Reichenow (1913) say that the male gametocyte of *Barrouxia schneideri* produces over 200 nuclei, at least four times as many as the maximum number produced by the schizont, and much the same may be said of the male gametocyte of *E. caviæ*. When, therefore, the male gametocyte is approaching maturity, its nuclei become very small and it is difficult to detect a definite karyosome in them. They appear to be equal in size; and their divisions appear to be more nearly synchronous than the early divisions are. It was in the nuclei of male gametocytes that the writer first thought he had seen structures resembling Tyzzer's (1929) illustrations of the peculiar mitoses he observed in the schizonts of *E. tenella,* but re-examination of these with higher magnifications convinced the writer that there was really no resemblance at all. No granules were present in any of the nuclei examined, although at first sight these appeared to be there. These nuclei of the male gametocyte are, indeed, so small that it is very difficult to determine exactly what happens when they divide. They frequently show the appearance depicted in Fig. 20, an appearance which resembles Fig. 19, Plate I, and text Fig. 3 in Léger and Duboscq's (1910) account of *Selenococcidium intermedium.* The subsequent stages in the development of the male gametocyte do not, however, resemble those of *Selenococcidium intermedium,* but are much more like those of *Barrouxia schneideri, Eimeria schubergi* and other typical coccidia.

At a stage later than this, the chromatin in these nuclei seems to take hæmatoxylin stains less deeply and to be distributed more generally round the periphery of each nucleus, so that gametocytes like the one drawn in Fig. 21 are seen. This probably represents the end of nuclear division, no more nuclei being produced before the male gametes are formed. The nuclei are all now

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at the surface and the chromatin in each nucleus collects, as Fig. 22 shows, along one side of the nuclear sphere, or, as Schellack and Reichenow (1913) put it in their description of the similar process in the formation of the male gametes of *Barrouxia schneideri,* it forms a cap over one part of the nuclear sphere. The sphere then elongates so that the chromatin cap is drawn out along one side of it and the succeeding phases are the transformation of this into the stumpy gametes, shown in Fig. 23, and the elongation (Figs. 24 and 25) of these into typical male gametes, composed almost entirely of chromatin. The male gametes are thus differentiated on the surface of the male gametocytes, as those of all the *Eimeriidea* (Wenyon, 1926) are. Sheather (1924) says that a small lightly stained area is visible at the anterior end of the gamete when it is stained with iron hæmatoxylin. The gametes have the two flagella characteristic of coccidian male gametes. Sheather (1924) states that these flagella arise on opposite sides of the curved, spindle-shaped body and are directed backwards, one being longer than the other. He gives their length as 6-9μ and that of the body of the gamete as 3μ. He was able to see in films male gametocytes from which the gametes had not yet been set free, so that their flagella were waving rhythmically all over the surface of the gametocyte. The writer has not been able to detect the flagella in sections. They are best seen in smear preparations.

The male gametes are, as usual, produced in great numbers and, when infestations are heavy, they are seen all over the sections and in masses in the lumina of the crypts of Lieberkühn. They fertilise the mature female gametes, which then immediately secrete a wall round themselves to exclude other male gametes and the development of the oocyst begins. Nothing like the fertilisation spindle of *Barrouxia schneideri* described by Schellack and Reichenow (1913) has been observed.

The Female Gametocyte and the Oocyst

In general the female gametocyte is an oval cell, the size of which varies, like that of the other phases, according to its age and the degree of its nutrition, which depends on the vigour of the strain of the parasite and the resistance of the host. The measurements made by the writer of a number of mature female gametocytes have been given in Part I.

The differences between the young female gametocyte and the young schizont, which it may closely resemble, have already been described. As development proceeds, the young female gametocyte elongates until a slender oval shape is attained, after which no marked increase in length occurs, but the accumulation in the cytoplasm of the granules described below results in a fattening of the transverse diameter, so that the mature female gametocyte (Figs. 29 and 30) has approximately the same shape as that of the oocyst. The male and female gametocytes do not, like the corresponding phases of some species of coccidia, differ strikingly in shape, nor is there a striking

difference in size between these two phases, like that shown by the male and female gametocytes of *Adelea ovata*.

When infestations are moderate, the female gametocytes are, like the other stages, most numerous in the cells lining the bases of the crypts of Lieberkühn; but when infestations are heavy, they may be found anywhere in the crypts. That it is possible for merozoites which become female gametocytes, and therefore develop into oocysts, to penetrate very exceptionally beyond the mucosa and even beyond the muscular layers of the wall of the intestine, is shown by the discovery, in one section only, of two mature oocysts just under the peritoneal layer. Male gametes must also have made the journey to fertilise these female gametocytes. The surrounding tissues were intact, so that distortion or breaking of the section could not have accidentally displaced these oocysts to their remarkable position.

The cytoplasm of the female gametocyte develops, at a very early stage in its growth, clusters of small spherical granules which, together with its single nucleus, distinguish it from the young schizont. As development proceeds, these granules increase in number and size, although some of them remain very small, even in the mature gametocyte. They stain black with iron hæmatoxylin and dark blue with Mayer's hæmalum or Delafield's hæmatoxylin. Many of them, especially the smaller ones, no doubt consist of the reserve food material which female gametocytes always contain; but the nature of others is revealed when they are stained with Heidenhain's azan or with Mallory's triple stain. The granules then take on brilliant colours, some staining yellow to orange, others red, these different colours indicating that the granules differ in chemical composition. In gametocytes whose size indicates that they are not full grown, either all the granules are coloured yellow with the azan stain or there are, amongst the yellow granules, a number of small or very small red granules, which later become as large as the yellow ones. The impression is thus gained that the yellow granules appear first and that the red ones are formed later, not by transformation of the yellow ones, but independently from the cytoplasm.

By the time that the female gametocytes are packed with these red and yellow granules (Fig. 29) they are brilliant objects. A little while later the granules which stain red with azan show a tendency to move to the periphery of the gametocyte and in older gametocytes these large red granules form a regular row all round the transverse section of the gametocyte, while its interior contains only granules which stain yellow, with at most a few scattered ones staining red. The two lowermost gametocytes in Fig. 30 show this arrangement of the granules; an attempt has been made in the figure to indicate, by differences in the tones of black and white, the colour contrasts brought out by the azan stain. It can also be seen in gametocytes stained with hæmatoxylin, but naturally the picture obtained is less striking with this stain. Throughout the development of the female gametocyte its nucleus appears,

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when it is stained with azan, as a homogenous body coloured violet to medium blue, with a bright red (or pink or orange according to the degree of staining) karyosome. With the same stain the cytoplasm of the female gametocyte throughout the development usually stains a faint blue if the slide has not been differentiated too far, but it is worth noting that, in sections in which the blue has all been differentiated out of the cytoplasm of the female gametocytes, the cytoplasm of the male gametocyte still retains a blue colour. It is not merely that the numerous granules in the female gametocytes obscure the blue in the cytoplasm, but that the cytoplasm of the male gametocytes stains a deeper blue and retains it longer against differentiation.

Female gametocytes at this stage may be regarded as mature and ready for fertilisation. Fertilisation has not been observed, but existing knowledge of the life history of coccidia shows that immediately it has occurred the gametocyte encloses itself in the first of its walls and thus excludes the entry of more than one male gamete.

The formation of this first oocyst wall is shown in Fig. 37 and the uppermost gametocyte drawn in Fig. 30. This latter gametocyte differs from the other two in Fig. 30 in two details of its structure. A wall is being formed round it in pieces which have a double contour and are as yet not united together; and its interior contains, as Heidenhain's azan stain clearly shows, yellow granules only, no red granules being usually seen at this stage or at most only a few small and scattered ones. It is evident that the disappearance of the red granules, which were arranged so strikingly in a regular row at the periphery, is correlated with the appearance of the pieces of the wall, which are, as the granules were, coloured red by the azan stain. Similar colour reactions are given by Mallory's triple stain and it is impossible to doubt that the substance of the red granules has gone to form the pieces of the first wall to be formed.

The process of the formation of the two walls of the oocyst from the granules in the cytoplasm of the female gametocyte was first described, so far as the writer is aware, by Simond (1897), in his study of *Eimeria stiedæ* (which was then called *Coccidium oviforme*) of the rabbit. Simond's account of the process and his figures of it in colour make the details clear. He stained his preparations with safranin and picro-indigo-carmine. Wasielewski (1904) confirmed his account from his own work on what is presumably the same species, which he calls *Eimeria cuniculi*. He notes that sometimes oocysts with imperfect walls are produced as a result, he thinks, of the action of pathological factors. Hadley (1911) has also described and clearly figured the conversion of the granules in the female gametocyte of "*Eimeria avium*" into the walls of its oocyst.

At a stage rather later than that shown in Figs. 30 and 37 the pieces of the oocyst wall have joined up to form a single complete outer wall (Fig. 31). The cytoplasm at this stage still contains many granules, but Heidenhain's azan and Mallory's triple stains show that the yellow granules now tend to

move outwards as the red ones did and to arrange themselves at the periphery of the fertilised gametocyte. They do this because their function is similar to that of the red granules. They form the inner wall of the oocysts, as Mallory's triple and Heidenhain's azan stains clearly show. With these stains the inner wall of the oocyst stains a yellow colour similar to that taken by the yellow granules and, when this inner wall has been formed, relatively very few granules of any kind remain inside the oocyst. Of those that do remain the majority stain yellow with the azan stain (Fig. 32) and doubtless represent surplus material not needed for the formation of the inner wall. They seem to degenerate, because the mature oocyst does not in sections (Fig. 33) usually contain any large granules at all. At most it shows very small granules peppered all over its cytoplasm, which doubtless represent reserve food. Only in overstained sections are larger granules visible and even these are not so large as the ones which form the walls and they are obviously artefacts. It has not been possible to determine whether the inner wall of the oocyst is first formed, as the outer wall is, in pieces, which later join together; such pieces have never been seen.

It is, of course, possible to follow all the details just described in sections which have been stained with any of the hæmatoxylins, but they are not then so evident. The granules which form the inner wall of the oocyst are not well stained by Mayer's hæmalum, but iron hæmatoxylin stains both kinds of granules very well.

The Oocyst

The general characteristics of the oocyst before sporulation are shown by Figs. 33 and 34. Fig. 34 is a photograph of freshly passed oocysts, and it gives some idea of the normal variations in size and shape, the larger oocysts being usually oval, the smaller ones either oval or almost spherical. The refractile contents are usually contracted well away from the innermost oocyst wall into a spherical or almost spherical mass. There is no micropyle. Henry (1932A) gives the thickness of the oocyst wall as 0.8μ and notes that it often has a brownish colour.

It is well known that it is difficult to get any fixative or stain to penetrate the walls of any coccidian oocyst evenly. For this reason oocysts in sections are frequently crumpled and distorted by fixatives into various irregular shapes, among which the halfmoon produced by invagination of one side into another is a common one. It is possible that the older oocysts are more liable to these distortions. For the same reasons the contents are often not well stained. This especially occurs when Heidenhain's azan and Mallory's triple stains are used, which involve rapid transference of sections from watery media to the higher grades of alcohol and back again. In well-fixed sections, however, they colour the cytoplasm of the oocyst various shades of blue. Sometimes the cytoplasm is yellow, a reaction which is possibly due to the presence in the

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cytoplasm of breakdown products of yellow granules which have not been used to form the inner wall. The sporulation of the oocyst and its nucleus have already been described. Fig. 35 is a photograph of the sporulated oocysts.

While the oocyst is inside the host the nucleus is always single, because sporulation does not begin until the oocyst has left the host in which it was formed. The contents then divide into the four sporoblasts, each containing two sporozoites, which are characteristic of all species of the *Eimeriinæ* (Wenyon, 1926). A residual body is usually by then visible near the centre of the oocyst.

Summary

A summary is given of existing knowledge of *E. caviæ*. It is a common parasite of guinea pigs, in England, Germany, America and probably also in other countries, but the presence of light or chronic infestations may be overlooked unless careful and successive daily examinations of the fæces for oocysts by an efficient flotation technique are made. It is especially difficult to say that a guinea pig has not had a previous infestation. Guinea pigs, in whose fæces no oocysts could be detected for several weeks, showed oocysts in the colon when sections of this were cut, so that they must have been carriers. It is suggested that, as a working compromise, at least six daily examinations should be made before a guinea pig is pronounced negative. On the other hand, in frozen sections, even if the material from which they are made has not been previously fixed, it is possible to detect gametocytes and oocysts. It is possible, therefore, by the use of frozen sections, to determine within two hours or less whether any oocysts being passed by a guinea pig are derived from an actual infestation or are merely passengers through its intestine. This method also eliminates the necessity of identifying any oocysts found in the fæces as those of *E. caviæ*.

When material is fixed and stained the necessity of comparing the results obtained with different combinations of fixatives and stains is emphasised.

The variations in the sizes of the different phases of the life history are discussed in relation to the suggestion that more than one species exists, and the conclusion is reached that these variations depend upon the host parasite relationship of different hosts with strains of the parasite of varying virulence and that they do not justify the creation of a second smaller species. No other facts are known that would justify the creation of a second species.

Experiments on the sporulation of the oocysts showed that this is not, at laboratory temperatures, appreciably quickened by aeration of the fluid in which it occurs, and that, at this temperature, it occurs at the same rate in water, 2 per cent. potassium bichromate, 1 per cent. formalin or a weak hypochlorite solution, the formation of the sporozoites in the sporoblasts in 60-70 per cent. of the oocysts requiring about eleven days. Other observers have

noted that sporulation is complete in 5-8 days at laboratory temperature or in three days in an incubator. Very few oocysts underwent sporulation after this period. Heavy infestations of resistant guinea pigs were produced by all these sporulated oocysts when they were fed to guinea pigs which had had previous infestations and neither the aeration nor the fluids used in which sporulation occurred made any difference to the degree of infestations. Investigation by physico-chemical methods of the factors governing the sporulation, correlated with studies by similar methods of the nature of the walls of the oocyst and of the liberation of the sporozoites from it, might lead to more efficient methods of control of all forms of coccidiosis. The cultivation of a coccidian species in a sterile medium would enable us to study the host parasite relationship more accurately and the writer hopes eventually to publish a method of doing this.

A summary of existing knowledge of the pathology and the clinical symptoms of the disease produced by *E. caviæ* is given. It produces little tissue reaction and often affects the host surprisingly little, perhaps because it usually attacks the colon only, although the cæcum may be lightly attacked. A resistance developed by the host, associated with allergic phenomena, has been described by Henry (1932).

The nucleus of the different phases of the life history is described. In general it resembles that of other species of coccidia, but always has a single karyosome and does not contain any rods or granules resembling chromosomes. It consists of a karyosome situated at one pole of an extrakaryosomatic part of the nucleus, both of these probably containing both plastin and chromatin. Around them is a clear zone of karyolymph which is not separated from the cytoplasm by a nuclear membrane.

The different and often puzzling appearances shown by the nucleus in preparations stained by different stains and differentiated to varying degrees are described. The division of the nucleus resembles that of the nucleus of many other coccidia and is not different in essentials from that of *E. schubergi*, described by Schaudinn. It is amitotic, no kinetic centres of any kind having been seen. The extrakaryosomatic part of the nucleus is withdrawn into the karyosome, which elongates till it has a dumb-bell shape and then divides into two parts, which reconstitute the form of the resting nucleus. Throughout the division the clear halo of karyolymph persists. Division of the nuclei of the schizont is frequently unequal, some of the nuclei dividing faster than others, so that the divisions are not synchronous.

The various phases of the life history are described and a description is given of the formation of the walls of the oocyst from the granules in the female gametocyte. The life history does not differ in any essential respect from that of *Eimeria schubergi*, so that *E. caviæ* could be used for teaching purposes. Almost every guinea pig is, or has been, infested with it, so that it is easily obtained; no other species is present in guinea pigs; and heavy in-

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festations, providing abundant material, can easily be obtained by feeding guinea pigs with oocysts, only the colon of these being required. It is thus a better source of teaching material than the centipede, *Lithobius forficatus*, which contains the four species, *Eimeria schubergi, E. lacazei, Adelea ovata* and *Barrouxia schneideri,* between which only the expert can differentiate. *E. caviæ* is big enough to present the student with little difficulty and yet small enough to exercise adequately his powers of observation.

Acknowledgments

The writer is greatly indebted to the Director of this Institute for the facilities which made this investigation possible and for his continual help and advice. M. O. J. McCarthy, M.R.C.V.S., constantly drew the writer's attention to interesting features in the large numbers of guinea pigs which he was daily examining. Without the constant help of the writer's laboratory assistant, H. E. Bowman, who looked after the guinea pigs, embedded and cut sections of the material examined and checked, with the writer, the oocyst counts and other technical details, the investigation would hardly have been possible.

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THE EPIZOOTIOLOGY OF FOWL CHOLERA

By J. F. CRAIG and A. RATTER

In this country outbreaks of fowl cholera are reported and recorded from time to time, but usually in isolated flocks. The annual number of outbreaks seen is small as compared with fowl typhoid or tuberculosis or even infectious laryngo-tracheitis, but the losses experienced in each of the outbreaks are often severe or at least appreciable. Perhaps the most serious outbreak in recent years was that encountered by Rice and referred to by him in 1926 where about 2,100 geese died out of 2,500 birds. But it is not uncommon to find 100 or 200 birds dying in an outbreak in a flock of four or five hundred.

According to the observations of Pritchett, Beaudette, and Hughes (1930), one may distinguish an epidemic and endemic form. The infection when present in endemic form varied with the season, increasing in amount and severity during the autumn months to a winter maximum and then declining during the summer season to a minimum. Furthermore, its prevalence and tendency to spread varied with the number of nasal carriers; a decrease in the carrier rate being accompanied by a decrease in disease mortality and in the occurrence of the local infections, roup, catarrh and œdema of the wattle. P. aviseptica infection in flocks of poultry with no history of previous cholera assumed the form of severe epidemic. The outbreaks arise during the winter months. The daily death rates were high; post-epidemic carriers among the survivors were uncommon. It was taken that in these two forms two separate strains of Pasteurella aviseptica could be identified by their cultural and other characters. The "blue" colony type was recovered from the endemic form, while the "fluorescent" colony type or an "intermediate" type was isolated from the epidemic form.

While unable to distinguish the types of organisms in the cases we have noted, the circumstances conform to the endemic and epidemic outbreaks described by these authors.

We have had an opportunity of studying a few outbreaks of fowl cholera in this area during the past few years. For the most part they have come under our notice during the months of September, October and November, when the outbreaks were at their height, but some losses had occurred prior to this time.

In two instances there was a history of fowl cholera having appeared in successive years, and in one of them there was very little doubt that some of the birds kept over from the previous outbreak had remained carriers, while in another case there was a possibility not only of carriers but also of infected ground.

It would appear that there is a tendency for the disease to die out in an outbreak. At least the losses reach a maximum and then they decline perhaps

because some resistance is acquired by some of the incontact birds. Certainly not all of the latter die of the affection. Moreover, towards the end of an outbreak the severity of the disease is not so great and catarrhal symptoms and œdema of the wattles are not uncommon. These facts support the view that the organisms may persist in some carriers in the nasal chambers.

The most severe losses are also inflicted after changes in weather conditions, such as a change to cold or wet weather.

In two outbreaks which came under observation it was the deaths among geese which attracted attention and it was evident that this was due to the fact that geese are highly susceptible and the disease in them very rapid and fatal.

The source of some outbreaks was not ascertained, but it was possible that fowl cholera was introduced by carriers or by visitors, by foodstuffs or food sacks.

In the control of this disease sanitary and disinfectant measures are readily put into practice, but the owner demurs against the destruction of many incontact birds and, of course, that may lead to the yearly recurrence of disease because of the presence of carriers. Vaccination, however effective, does not appear to us to give a lead in preventing the presence of carriers or the spread of disease when flocks are sold. We have found in more than one outbreak that the extension of the disease was avoided by keeping the survivors under intensive conditions in one cabin until the end of the laying season. After that they were disposed of for food.

There is some need for control of the indiscriminate sale of adult fowls, the use to which such birds are put, and of the employment of food sacks for carrying dead birds.

Here are the details of some of the outbreaks which have come under our observation in this area.

Outbreak A was seen on September 5, 1936. The owner had 2,000 fowls which were bought as pullets in January and February of the same year. They were housed in eight separate cabins and were given free range, free range being given for each pair of cabins. The fencing was good between the different pairs or lots of cabins. The outbreak began about four weeks previously and was confined to two cabins having the same free range. Sixty birds had died in one of these cabins and 20 in another. The history was that the birds would appear in perfect health when seen at night, and next morning would be found dead. This state of affairs continued for two weeks and then a catarrhal affection appeared in some of the remaining birds in both cabins. The birds had a catarrhal discharge from the nares and eyes, and lingered for a day or two and then died.

In the other cabins the fowls remained healthy. By September 11 only two birds with catarrh were discovered in the two diseased cabins. The other birds were in their normal health. No evidence was obtainable of a previous

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attack on the place and the only probable explanations that could be given of the source of infection were: (1) that some of the pullets bought in January and February were carriers, but that did not seem feasible in view of the confinement of the disease to the two cabins, and (2) that it was brought in with food, sacks or other material. No losses were discovered as occurring recently in the neighbourhood. In the early days of the outbreak the owner suspected quicklime poisoning.

Two live birds were brought in on September 5 and were killed, but did not show anything more than suspicious features.

On September 8, one dead and three live birds were brought in for examination.

In the dead pullet the lesions consisted of small hæmorrhages in the peritoneal fat and overlying muscles, some enteritis and a very fatty liver. Pasteurellæ were numerous in blood films and a pigeon inoculated with heart blood died the following day with numerous bipolar organisms in the blood.

On the 9th one of the live birds had died, while the other two were moping, hardly able to stand and lay with their heads down or turned round to one side. Their temperatures were 105 and 108 deg. F. Droppings were watery, yellow and stinking.

Lesions.—Liver light brown, enlarged, studded with yellow-white round necrotic areas, a thin fibrinous layer on the liver and pericardium and considerable quantity of yellow fibrin over ovary and intestines, some enteritis, a catarrhal discharge from the nares, a little serous fluid in the pericardium, left lung solidified and covered with some fibrin, kidneys yellow and soft. Films from the solid lung contained numerous bipolar organisms and a few were also noted in the blood.

One of the two live birds died on the 14th. The carcass was very poor and head pale, tissues moist, lungs œdematous, some fibrinous flakes over gizzard and small hæmorrhages on mucous membrane of proventriculus as well as enteritis in anterior portion of bowel. There were numerous coccidia in this part of the bowel. Blood films contained a few pasteurella.

The last bird recovered.

At the end of the outbreak two birds, one affected with catarrh of the nares and eyes, the only diseased birds on the place, were brought for examination. One of the latter died on September 14. The chief lesions were some light patches in the liver, deposit of fibrin over the ovary and intestines and enteritis in the anterior part of the latter with bloodstained ingesta. The bird with what appeared like catarrhal roup died on the 16th and very few changes were noted except those in the nasal chambers and eyes. Cultures and films from the heart, blood and eyes contained pasteurella.

Two healthy birds inoculated from the nasal and eye discharges of the latter bird died in the course of ten days with lesions of enteritis and serous pericarditis. Pasteurella were isolated from the blood.

It would thus appear that in the latter stages of the outbreak nasal catarrhal symptoms were caused by pasteurella infection.

The organisms in these cases presented the usual morphological and cultural characters as well as the pathogenic properties of pasteurella. They produced acid, but no gas in mannite and saccharose and very slight acid change in dextrose.

The action taken in this outbreak consisted in the attention to the proper disposal of the litter and thorough disinfection of the cabins and the application of quicklime to the land. The disease did not spread to any of the other cabins. In each of the cabins where the outbreak occurred 60 birds were left, which remained healthy. No further trouble occurred on this place. It would not appear therefore that any of the birds remained as carriers.

Outbreak B.—Another outbreak was investigated in October, 1936. The owner stated that he had losses from fowl cholera yearly for six years. He kept usually about 800 fowls on 102 acres of land and lost every year about 200 of them. This year the first death occurred in August and by October, 94 had died, one of them showing œdema of the wattles. The disease was confined to pullets. These birds were kept in two night arks which were near to one another and which had been kept vacant before use for four months after thorough disinfection and dressing of the floors with quicklime. The pullets had been hatched out and reared on the place. It did not appear that the birds carried the infection into the huts. Suspicion fell upon 140 ducks which were kept on ground near to the infected huts, but among them there was no sickness. As the outbreak wore itself out, 135 birds remained in apparent good health in the two huts which were implicated. Apparently on this place the infection remained from year to year, possibly in nasal chambers of carriers or on the ground. But apart from disinfection, the owner was not prepared to dispose of the 135 birds from the affected huts, although some of them may become carriers. The birds were now kept on the intensive system.

Outbreak C.—This outbreak was brought to our notice on November 2, 1936. The owners were dealers in poultry and had a small poultry farm on which they kept fowls during the course of trading. The number of fowls on the place varied a good deal up to a few hundreds. Some geese were also reared. All the birds were on free range. Twenty-six geese were bought as goslings about the middle of June of that year. Two deaths among the geese occurred early in October, but within the last few days 12 to 15 died suddenly. Six were found dead on November 1 and five died the following day. Two dead geese were examined and the lesions were identical in both cases. They were hæmorrhages under the epicardium and endocardium, an intense hæmorrhagic enteritis, the posterior bowel being full of blood and blood clots, numerous pinpoint white spots on the liver and congestion of the

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lungs. Blood films were rich in pasteurella and cultural and other characters were typical of these organisms.

Subcutaneous inoculation of two fowls with cultures caused death in 24 hours. In both of them there were necrotic foci in the liver, small hæmorrhages under the epicardium and an intense hæmorrhagic enteritis. The pasteurella were identified in the heart and blood and liver.

It was only ascertained in conversation that the hens were allowed to run at grass with the geese and that approximately 60 birds were lost within the last six months, but no attention was paid to these losses until the geese began to die and even then the connection between them was not realised. Some two or three dozen chickens only were left and one dozen were lost in the last fortnight. The fowls were bought from all sorts of sources.

It was requested that some dead fowls should be forwarded in order to determine whether they were dying from the same cause as the geese. Two moribund fowls were brought in on November 4 and examined post-mortem. In one there were hæmorrhages on the epicardium along the coronary groove and pneumonia and in the other seri-fibrinous pericarditis and pneumonia. Pasteurella were identified in the blood and lungs and isolated in culture.

On our advice the remaining live healthy birds were destroyed and sold for table use, the houses, etc., disinfected, a pool on the land drained and the land dressed with quicklime in the proportion of four tons to the acre. The place was to be kept vacant of poultry for some months.

The owner had never had losses among his geese in previous years and the cause of any losses among the poultry had not previously been investigated. In view of the absence of mortality in his geese hitherto, it was concluded that the disease had been introduced by the poultry purchased from time to time.

Outbreak D.—The outbreak in this poultry farm started in June, 1938, and the deaths up to October 24, 1938, amounted to 100 fowls out of a total of 500 birds. The birds were on free range and housed in three large cabins, in all of which the disease had appeared. The owner had this disease yearly for a few years and in 1937 he lost 200 birds out of a total of 400; 130 birds now remained out of the previous year's outbreak. Four live birds were still showing evidence of illness, two being affected with nasal catarrh. Of these, three died and were found to be infected with fowl cholera, one being also affected with extensive tuberculosis.

In addition to the catarrh there were found in all cases at the post-mortem examinations some enteritis, hæmorrhages on the epicardium and fibrinous deposits over the ovary and intestines. Pasteurella were identified in all cases in the blood and isolated in culture. All the remaining birds were afterwards kept on the intensive system after due attention to disinfection and were retained until the laying season had terminated.

It would seem in this outbreak that some of the 130 birds from the previous year's outbreak were carriers and the disease had been set going again by some debilitating circumstances. In view of the catarrhal cases when the disease was apparently wearing itself out, it is not improbable that the infection in some of the light or suppressed cases may have been retained in the nasal passages as saprophytic organisms until conditions became favourable for the invasion of the body and the production and spread of disease.

Outbreak E.—The last outbreak came under our notice on October 27, 1939. Here again the geese were first noted to be affected. Ten geese had died and these were hatched on the farm from eggs purchased from another local farmer.

The geese were mixed with the hens and fed with them. The hens showed no signs of ill-health until October 31 when four pullets died. These birds had been kept on ground which was very hard and dry until the recent rains which converted the ground into puddles much used by the geese. Then the losses appeared. The number of fowls on the place only amounted to about 50.

One dead goose and one pullet were examined post-mortem.

In the goose the chief lesions were some petechiæ on the epicardium and enteritis and a few amidostoma in the gizzard. Numerous pasteurella were present in blood films and isolated in culture. A rabbit and a pigeon inoculated subcutaneously from a culture died in 24 hours and numerous pasteurella were found in blood films.

In the pullet the comb and mucous membrane of the mouth and pharynx were purple, the liver dotted with yellow-white spots, the lungs solid and œdematous, the epicardium dotted with petechiæ and the anterior portion of the intestines affected with hæmorrhagic enteritis.

Numerous bipolar organisms were present in the blood and isolated in culture.

In what way the disease entered this flock is not apparent. There was not known to have been a previous outbreak; it is not likely that the disease was brought in by the eggs of the geese. But it is curious that the geese appeared to be affected first; certainly they seemed to be highly susceptible. The fowls were chiefly bred on the place. One can only suppose that fowl cholera was introduced with the food, sacks or persons rather than through the medium of carriers.

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A CASE OF PYOMETRITIS

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THE subject is a maiden Dalmatian, nearly 11 years and 6 months at the time of the external manifestation of the condition.

History.—In good health and condition up to July, 1939, when her appetite became fickle, her breath unpleasant and at times inquiries were made as to whether she was in whelp. There was no record of an opportunity for mating. On examination there was no definite evidence of pregnancy and I suspected pyometritis. I advised waiting developments. The bitch remained in good health and spirits until about the end of October when her breath became more unpleasant, rather more distension of the abdomen, although the time for whelping had long gone by; there was evidence of discomfort at times, occasional vomition and little or no inclination for exercise. On or about November 20 a slight purulent vaginal discharge was noticed which not only increased in quantity but contained purple streaks of pus. The discharge was so great and objectionable that it was necessary to mop up after her when she crossed the floor. Temperature 104 deg. F.

Diagnosis.—Pyometritis.

Prognosis.—Not too favourable.

Treatment.—Operation advised without delay, but the owner objected to it until all else had failed. I gave little or no hope of recovery by resorting to medicinal measures. However, I decided to try antibacsyn and injected 4 c.c. The following day the temperature had fallen to 102.5 and pus was literally pouring from the vagina. Another 5 c.c. of antibacsyn was injected and the vagina irrigated with weak antiseptic solution. On the third day the purplish character of the pus was more pronounced, but there was less discharge. Temperature 101. On the fourth day there was very little pus (which was not so fœtid), the breath was less smelly and the patient was nearer her normal self. Five c.c. antibacsyn injected. By the sixth day the discharge had completely disappeared. There has been no recurrence to date and the bitch is apparently in the best of health, condition and spirits.

Reviews

Transactions of the Omsk Veterinary Institute. Vol. II. Published by the *Omskia Pravda,* Omsk, 1938. Pp. 235.

THE book under review consists of twenty different articles dealing with many aspects of veterinary science. The work reported in this volume has

been done by the members of the various departments of the Omsk Institute, and thirty-three authors are responsible for the completion of it.

The subjects chosen are varied and can be divided into three groups:

1. Those of academic interest only.

2. Those of importance to Russian workers, particularly of the Omsk and its surrounding districts.

3. Those of general interest.

The Department of Histology investigated the structures of the skins of local Siberian sheep. It was found that the literature on the subject was not extensive and its importance cannot be under-estimated in breeding of sheep. The writers of this work undertook a series of analyses of skins of one special breed of Siberian sheep. This was carried further by crossing this breed with others and ascertaining what qualities in the structures of the skins are transmitted to the resultant progeny.

The texture of wools was also investigated and a special article is devoted to various types of wool specially suitable for making felt boots. Another article from the same department is on the structure of the lips of Northern deer (Rangifer Terandus). Both anatomical and histological examinations are reported and comparison is made to the structures of lips of other breeds of deer and domesticated animals.

Several articles are of special interest to Russian workers. These include:

1. Watering places in the breeding locality of the Omsk district—these appear to be in need of improvement, as most are rather dirty and unpalatable even to animals.

2. Feeding of Northern deer.

3. Infective pleuro-pneumonia of goats of Altai district.

4. Rearing of a special breed of goats whose wool is suitable for making felt boots that are so commonly used in Russia.

5. Common tape worms infesting Northern deer. These are of the Anoplocephalata group, and the two types most prevalent are:

(a) Monieza Cæri (scriabin).

(b) Monieza expansa.

The writers strongly recommend to all Soviet workers to deal with the problem by strict attention to the administration of vermifuges in the autumn before the deer are sent out on to the winter pastures, frequent changes of which are most essential.

The helminthology of three groups of dogs and treatment by carbon tetrachloride occupies a prominent position in this volume. The animals examined are in *three* groups:

1. Dogs in sheep-rearing district.

2. Dogs on farms.

3. Town stray dogs.

Altogether 64 post-mortem examinations were made.

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TABLE I

Helminthological flora of country dogs of Omsk and district.

Nematodes	Total No. investigated	No. infected	% infected
1. Toxocara canis	64	42	65
2. Toxascaris leonina	„	12	18
3. Uncinarina stenocephala ...	„	6	9
Cestodes			
4. T. hydatigena	„	9	13.5
5. T. senata (pisiformis) ...	„	1	1.5
6. Echinococcus granulosus ...	„	11	17
7. Dipylidium caninum ...	„	29	45
8. Mesocestoides lineatus ...	„	16	25
Trematodes			
9. Opisthorchis felineus ...	„	9	13.5
10. Alaria-alata	„	1	1.5

TABLE II

Helminthological flora of dogs inhabiting farms and being in contact with all farm animals.

Several collective farms were investigated and the resultant sum of infection is seen below.

Parasites	Total No. investigated	No. infected	% of infected
Flat worms			
1. T. hydatigena	16	10	62.50
2. T. pisiformis	„	1	6.25
3. Multiceps multiceps ...	„	4	25
4. D. caninum	„	7	43.75
5. Mesocestoides lineatus ...	„	1	6.25
Round worms			
6. Toxascaris leonina ...	„	11	68.7
7. Toxocara canis	„	4	6.25
8. Uncinaria stenocephala ...	„	7	43.75

Vermifuges of dogs, according to these authors, are not clearly worked out. In an article dealing with this subject it is reported that carbon tetrachloride (CCl_4) is very toxic to sheep, deer and the bovine species, and that dogs are capable of tolerating fairly large doses. Carbon tetrachloride is of some value in the treatment of ascaris and ancylostomida. It has no action on immature forms of ascaris, or on the cestode group. The full toxicity of this drug in dogs is not worked out, and caution is necessary particularly in

cases where there may be a deficiency of calcium in the blood. Dose recommended is .2-.3 c.c. per kilo. body weight, mixed in 15-30 grs. ol. ricini. Therapeutic value is enhanced by previous starvation of the patient for 12-24 hours.

Another drug reported on is calomel—particularly in the treatment of diarrhœa in calves. This was based on the knowledge of using it in the treatment of diarrhœa in young children. The author found that calomel as an intestinal antiseptic is not irritating to the mucous membranes of the gastro-intestinal tract. It increases secretions of the intestinal wall due to irritation of the nerve endings of parasympathetic system and Auerbach's plexus. The chief facts of value have been found to be: (a) highly antiseptic; (b) able to increase intestinal secretions and peristalsis; (c) negligible degree of absorption. Another drug used in the above disease is Rivanol. It is particularly useful in dietetic cases in young calves.

Further work on the bovine species reported is that of finding the influence of various factors on the agglutination titre in brucellosis infection. In determining the agglutination titre several essential points must be considered in order to arrive at the true value of the titre. It was found that duration of pregnancy, calving, and milking all tend to change the titre. Milking tends to lower the titre; several experiments have shown that feeding has no influence on the titre. In all the experimental animals used it was found that the agglutination titre is markedly changed in the last stages of pregnancy. Before parturition it gradually rises, but two or three days after it either decreases or a positive titre becomes doubtful or more often negative.

There is only one surgical case reported and is that of abdominal hernia in a calf (male) of six weeks old. The case is of particular interest due to several abnormalities—the sacrum was composed of four vertebræ, and there appeared to be a complete absence of caudal vertebræ. Aponeurosis of abdominal muscles along the white line was not present; as a result the abdominal organs were found in an extensive hernial sac. The operation consisted of reducing and removing the hernial sac and creation of a new artificial white line. Anæsthesia was induced by intravenous injection of physiological solution of NaCl (lege artis), glucose and spirit vini. A new white line was created by suturing the muscle layers with catgut and silk sutures were used in the skin. The scar was covered with collodion. Recovery was uneventful. The superficial sutures were removed after eight days; examination of the patient 15 days after the operation revealed a strong newly formed white line.

The Department of Poultry reported two cases, one on air sacs of poultry and the other on ultra-violet ray exposure of incubator-bred chickens. These chicks were found to be more prone to rickets due to marked avitaminosis. The essential vitamin D, derived from the liver of cod, cannot be obtained in sufficiently large supplies to correct the rachitic lesions and the

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Soviet workers have to resort to the use of ultra-violet ray of the chicks and the subsequent feeding of chicks with the irradiated yeast. The irradiation was used both as preventive and curative means of combating rickets. The treatment in well-developed lesions was effective in 54 per cent. cases.

The general impression of this work is that a great deal is being attempted but the need for further research is emphasised in most of the sections by the various authors.

The Principles of Parasite Control. By THOMAS W. M. CAMERON. Pp. 48, Diagrams 3. Published by National Business Publication, Ltd., Gardenvale, Quebec, Canada. Price 50 cents in Canada, 2s. 6d. in Great Britain. Date of publication, April 30, 1940.

PROFESSOR CAMERON'S new book is one of those small volumes which prove such treasured additions, both to the general practitioner's and specialist's bookshelves. The book is attractively printed and bound, written in a style which holds the reader's interest and contains a vast store of practical information. Anyone who has given thought at all to the intelligent and practical control of parasitic diseases in man or animals will find this work a help and inspiration. Not only are the writer's viewpoints incorporated in the text, but also most of the principles underlying the control of parasites which have been expressed by numerous workers in many textbooks and journals, published over a period of years. When such principles are collected and logically arranged in a monograph, the advantages of access to such a work need no further elucidation.

The introductory part of the book deals largely with significant facts regarding bionomics; discussions are illustrated frequently by helpful examples of specific forms of parasitism in various animals. The second half of the text is concerned with the principles of control. The treatment and disposal of manure is given considerable space; the usefulness of over one hundred chemicals tested for destroying helminth ova and larvæ is tabulated. The value of various chemical, physical, sanitary and hygienic methods of controlling parasitism are discussed under practically every sub-heading in which those concerned in the control of parasites may require information.

AUGUST NUMBER

Because of the necessity for economy in the use of paper it has been decided not to publish an August issue of THE VETERINARY JOURNAL, but to publish a combined August/September number which will be enlarged.